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Pseudotumour cerebri
Idiopathic intracranial hypertension and vascular intracranial hypertension

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Abstract: From the first to use of "*pseudotumor cerebri*" by Nonne in 1904, the historic evolution of the knowledge on pseudotumor cerebri has been marked by several periods (the otologic stage, the neurosurgical stage, the neuro-ophthalmologic stage); today there are clear diagnosis criteria for the idiopathic intracranial hypertension, there is a clear differentiation between idiopathic intracranial hypertension and vascular intracranial hypertension, also the comprehension of the illness pathogeny is based on the dynamics of the intracranial fluids, which allows the auto-regulation of the cerebral circulation within quasi-normal limits, despite the very high intracranial pressure.
Key words: idiopathic intracranial hypertension, pseudotumor cerebri, vascular intracranial hypertension

Vascular intracranial hypertension

Intracranial hypertension can occur in cerebral-vascular illnesses due to sanguine, cerebral or extra-cerebral, circulatory disorders, which modify the dynamics of the intracranial fluids and cause the intracranial pressure increase. There are disorders in the auto-regulation of the cerebral hemodynamics and the cerebral parenchyma volume continues to increase due to the brain edema or to the increase in the cerebral sanguine volume (brain swelling) with the secondary increase in the intracranial pressure.

The volume of the cerebral parenchyma increases due to the modifications occurred at the level of the cerebral sanguine capillaries, which leads to:

- the occurrence of the extracellular brain edema due to the increased quantity of interstitial fluid:
- extracellular edema produced by a hydrostatic mechanism (ultra-filtration) in severe arterial hypertension,
- extracellular edema with oncotic induction (vasogenic edema) due to an increased permeability of the brain blood

barrier (open brain-blood barrier)

- cerebral congestive edematization with an increase in the volume of the cerebral parenchyma by vascular dilatation.

The vascular types of intracranial hypertension have characteristic etiologies and they occur by:

- slowing down or decreasing the intracranial venous flux in thrombophlebites and cerebral venous thrombosis, the decrease in the venous flux at the level of the superior longitudinal sinus (SLS) directly in compressive lesions (hollowing fracture, etc.) or in SLS shunting by an intracranial arterial-venous malformation, or the extra-cranial illnesses that block the returning venous circulation at the cervical level, reduce the cerebral venous drainage and cause the decrease in the absorption of the cranial-spinal fluid and then the occurrence of the brain edema.

- in hypertensive encephalopathy, when the hydrostatic brain edema occurs (by ultra-filtration), as well as a brain swelling (by vasodilatation).

- the cerebral ischemia or the ischemic stroke reduces the arterial sanguine contribution and causes an ischemic brain edema, which is a mixed brain edema, both a cellular edema (cytotoxic) and an extracellular brain edema with oncotic induction (vasogenic).

Cerebral venous thrombosis

Cerebral venous thrombosis reduce the returning venous circulation from the brain and the skull, a venous stasis is produced and the cerebral sanguine circulation is slowed down. There are areas of cerebral hypo-anorexia concomitantly to areas of venous congestive edematization, and the cellular cerebral (cytotoxic) edema occurs, as well as the oncotic extracellular (vasogenic) edema, which evolves to a mixed brain edema. The venous sinuses also ensure the resorption of the cerebrospinal fluid, and the thrombosis of the venous sinuses leads to a diminished drainage of the cerebrospinal fluid. Therefore, a progressive intra-ventricular accumulation of cerebrospinal fluid occurs, with a pressure increase in the ventricular system and the occurrence of the hydrocephalic brain edema. These phenomena happen slowly, in varied successions, but the evolution is progressive towards an intracranial pressure increase.

The iatrogenic thrombosis of the internal jugular veins is quoted in cases of prolonged use of the jugular catheters for the intravenous administration of medication. In such cases, the same pathogenic processes occur, and the ICH syndrome may appear.

The symptomatology is caused by the initial causal lesion, after which neurological focal symptoms may occur related to the progression of the venous thrombosis, as well as symptoms caused by the intracranial pressure increase. A venous infarct often happens, which is associated to a cerebral hemorrhage, which also aggravates the neurological clinical presentation.

Usually, the clinic evolves to an incomplete or complete syndrome of intracranial hypertension.

The main characteristics of the intracranial pressure increase in cerebral venous thrombosis are:

- a slow increase in the intracranial

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pressure up to the normal limit value of 20 mm Hg, usually during a period of a few days,

- over the value of 20 mm Hg, the ICP increase continues to be progressive, and it may reach maximum values of approximately 30 mm Hg in a few hours or days.

This gradual increase allows the compensating mechanisms to act more efficiently, and also for the applied therapy to encourage the intracranial pressure decrease and the improvement of the cerebral sanguine circulation.

- the maximum values that may be reached in cases of intracranial hypertension syndrome are of approximately 30- 35 mm Hg (sometimes the maximum values may be of 40 mm Hg) and

- the pathological pressure values may last for several weeks, with a slow return to normal pressure values and period of intermittent increases,

- usually, there is a recurrence to intracranial pressure values of about and above 20 mm Hg, which causes the persistency of a prolonged attenuated symptomatology.

The treatment of the venous thrombosis with an ICH syndrome is:

- etiological and pathogenic for the vascular disorder, when possible,

- pathogenic for the intracranial hypertension syndrome.

A particular pathogenic mechanism is the reduction of the venous flux at the level of the superior longitudinal sinus (SLS) with an important blockage of the CSF resorption:

- in the case of arterial-venous malformations of the Galien vein (Galien vein aneurisms), when there is a SLS shunting by malformation and the cerebrospinal fluid resorption is diminished by the occurrence of the hydrocephalus. In infants and small children, the dominating symptomatology is the cardiac disorder due to the increased venous return, while the ICH syndrome also occurs in older children.

- in the case of a median intrusive cranial fracture, which interests the third posterior part of the SLS.

- in children with craniostenoses, there may be anomalies of the venous drainage, which interests the sigmoid sinus and the jugular vein, which may cause a venous hypertension, with a diminished drainage of the cerebrospinal fluid and the increase in the intracranial pressure. Usually, the phenomenon occurs up to the age of 6 years old, after which a collateral venous drainage is developed by the stylomastoid plexus, leading to the normalization of the intracranial pressure.

Hypertensive encephalopathy

High blood pressure is the most important predisposing factor for cerebral-vascular illnesses, and the most frequent complication is the cerebral hemorrhage. The exaggerated increase in the values of the systemic blood pressure also causes disorders of the cerebral circulation auto-regulation, with other secondary cerebral suffering.

Hypertensive encephalopathy is defined in the clinical presentation of induced intracranial hypertension by an acute episode of arterial hypertension.

1. The acute hypertensive encephalopathy is caused by the acute blood pressure increase in:

- severe high blood pressure,
- uncontrolled / untreated high blood pressurein pregnancy (eclampsia),
- high blood pressure in glomerulonephritis, pheochromocytoma, etc.

The acute increase in the sanguine pressure values leads to the inefficiency of the cerebral vascular auto-regulation, a generalized cerebral vascular dilatation occurs and / or there is an increased permeability of the cerebral capillaries. The increased permeability in the brain blood barrier has been constant more frequently at the level of the gray matter.

Therefore, the increase in the volume of the cerebral parenchyma is caused by:

- brain swelling by vasodilatation,
- hydrostatic extracellular brain edema, by ultra-filtration when the brain blood barrier is intact (close brain-blood barrier)
- oncotic (vasogenic) extracellular brain edema by an injury of the brain blood barrier (open brain-blood barrier).

The posterior reversible encephalopathy syndrome (PRES) or the reversible posterior leuco-encephalopathy syndrome (RPLS) with a hypertensive etiology is included in the acute form of vascular etiopathogeny ICH. The clinical presentationis typical and the DWI exploration shows an extracellular brain edema by the increase in the water mobility with a posterior bilateral location, and a sub-cortical interest in the white matter too. The treatment consists of decreasing the systemic blood pressure.

2. The chronic hypertensive encephalopathy (Binswanger encephalopathy) is a rare cerebral-vascular illness with a chronic extracellular brain edema: hydrostatic brain edema combined with the oncotic brain edema.

The intracranial pressure increase in acute hypertensive encephalopathies are characterized by:

- the relatively high speed with which the intracranial pressure reaches the normal threshold value in approximately a few hours
- the ICP continues to increase above the normal values for a period that is usually shorter than the previous interval, of few hours only
- the maximum values that the ICP may reach are of 30 – 50 mm Hg and
- the period with pathologic intracranial pressure values is usually of several hours, rarely of several days. The anti-hypertensive treatment improves the clinical condition.

The unmonitored hypertensive patients, or those who are incompletely treated may present repeated episodes of hypertensive encephalopathy, or they may suffer from the most frequent complication, which is the cerebral hemorrhage.

The clinical evolution of hypertensive encephalopathy is up to an incomplete syndrome of intracranial hypertension, and it has a regressive aspect. In the pathogeny of the syndrome, there is an auto-limiting mechanism: the intracranial pressure increase caused by the increase in the sanguine blood pressure and by occurrence of the cerebral vasodilatation generated the collapse of the walls of the intracranial sanguine vessels, and, to a certain extent, to a diminished cerebral sanguine volume. The mechanism consists of the direct action of the increased intracranial

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pressure over a functional disorder that is secondary to the exceeded auto-regulation of the cerebral circulation, and it has a limited value.

The treatment of the hypertensive encephalopathy is both etiologic and pathogenic:

- the treatment of the hypertensive episode, as an etiologic aspect, and

- the pathogenic treatment of the intracranial hypertension syndrome.

Ischaemic stroke

The ischemic stroke represents 85 % of the cerebral-vascular illnesses. The large ischemic cerebral lesions are accompanied by the brain edema with cerebral herniation (sub-falciform) and by the intracranial pressure increase.

The extended cerebral ischemic infarct with phenomena of intracranial hypertension is caused by the occlusion or stenosis of a great cerebral artery: the internal carotid artery or a terminal branch that irrigates a vast territory, such as the middle cerebral artery. The extended ischemic infarct of the Sylvian artery occurs in approximately 10 % of the patients with acute cerebral circulatory insufficiency, and it has been designated as the malign infarct of the Sylvian artery due to the increased mortality, of up to 80 % of cases, despite the therapeutic means used. The massive cerebellum ischemic infarct can cause the collapse of the 4th ventricle with the occurrence of an acute obstructive hydrocephalus and an acute ICH syndrome, and it has a direct compressive effect on the brainstem with the manifestation of vegetative disorders.

In the case of the cerebral hemispheric ischemic stroke, the decreased sanguine flow in the territory of the middle cerebral artery leads to the occurrence of certain ischemic metabolic disorders at the level of the affected cerebral parenchyma. The permeability of the cerebral capillaries increases (open brain-blood barrier) and the extracellular oncotic (vasogenic) edema occurs. The evolution is usually a rapid one, with the extension of the brain edema, the increase in the intracranial pressure and the occurrence of the sub-falciform cerebral hernia (median line movement towards the unaffected cerebral hemisphere). Although the intracranial pressure increasing mechanism is based on the cerebral ischemia with a hypoxic brain edema, which is characteristic for the parenchymatous lesions, while the etiology is represented by the impacts on a great cerebral artery, and it includes the ischemic stroke on vascular intracranial hypertension.

Since the moment of the arterial occlusion, the intracranial pressure increase is:

- rapid until it reaches the normal pressure limit of 20 mm Hg, by the progression of the brain edema and the surpassing of the pressure compensating possibilities, with a duration of up to several hours

- above the normal pressure values, the ICP increase is also a rapid one, and the maximum values are reached within a short interval of time: half an hour – several hours

- the maximum values of the intracranial pressure are of approximately 40 – 50 mm Hg and

- the duration of these pathological values is of several days and it corresponds to the

intensive care period.

The evolution is rapid towards the decompensation of the intracranial hypertension with almost 80% unfavorable results despite the applied treatments. There is an attempt in using the etiological treatment of the arterial obstruction and the pathogenic treatment for the ICH syndrome. During the first three hours from the beginning, there may be an intravenous administration of recombined tissue plasminogen activator (rtPA) in a dose of 0.9 mg/kg, maximum 90 mg; the administration of streptokinase or of other thrombolytic agents does not have the same efficiency as rtPA. The brain edema receives a pathogenic treatment with osmotic diuretics (mannitol), and hyperventilation if there is an imminent decompensation of intracranial hypertension and the production of a brain herniation etc.

Sometimes, there is an attempt of a surgical intervention:

- decompressive craniectomy of posterior cerebral fosse and of evacuation of a cerebellum infarct with a compressive effect on the brainstem, perhaps with a ventricular drainage,

- decompressive craniectomy and the evacuation of a cerebral hemispheric massive infarct, which may diminish the intracranial hypertension, but the surviving patients is left with major neurological deficits.

A particular case of generalized cerebral ischemia is met in the post-resuscitation syndrome when the sanguine flux disorder includes the entire brain, with a complete ischemia throughout the stroke, followed by reperfusion disorders.

The consequence of this cerebral circulatory failure, primary – before and during cardiopulmonary resuscitation, and secondary ischemic damage, during reperfusion is the development of the mixed brain edema: both cytotoxic and vasogenic, concomitantly to the production of the glial-neuronal necrosis. The hyperemic reperfusion may exacerbate the brain edema. The mixed brain edema accentuates the elevated intracranial pressure and it exacerbates the brain injury. The treatment is complex and the results do not compensate the efforts.

TABLE I

Etio-pathogenic and evolutionary characteristics of the various forms of vascular ICH

Cerebral venous thrombosis	**Hypertensive encephalopathy**	**Ischemic stroke**
Cerebral vascular pathology: - thrombosis of dural sinuses - thrombosis of cortical veins	Cerebral vascular pathology: - dilatation of cerebral arteries	Cerebral vascular pathology: - infarct of Sylvian artery - massive cerebellum infarct
Cerebral blood flow : Reduced venous outflow	Cerebral blood flow : Increase arterial inflow	Cerebral blood flow : Reduced arterial inflow
Pathogenesis: - venous dilatation; open BBB and vasogenic brain edema and	Pathogenesis: - dilatation of cerebral vessels; closed BBB and hydrostatic brain edema, and	Pathogenesis: - ischemic increased capillary permeability ; open BBB and vasogenic brain edema

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- diminished CSF drainage with hydrocephalic brain edema	-increased vascular permeability with open BBB and vasogenic brain edema	
ICP increase: - slow to the normal limit - slow above the normal limit	ICP increase: - rapid to the normal limit - slow above the normal limit	ICP increase: - rapid to the normal limit - rapid above the normal limit
Sub-acute and chronic evolution Possible decompensation	Acute and sub-acute evolution Rarely decompensation	Acute evolution Usually decompensation
Pathogenic treatment	Pathogenic and etiologic treatment	Etiologic and pathogenic treatment, Decompressive craniectomy

Idiopathic intracranial hypertension

Idiopathic intracranial hypertension is a syndrome characterized by the intracranial pressure increase in the absence of an expansive intracranial process, of hydrocephalus, of an intracranial infection, of dural venous sinus thrombosis, of hypertensive encephalopathy and without a neurotoxic etiology. The idiopathic intracranial hypertension partially corresponds to the old designation of pseudotumor cerebri; the term of benign intracranial hypertension has also been used, but it was subsequently dropped as it was noticed that the evolution of this illness can be accompanied by complications that exclude the idea of benignity (visual acuity decrease to cecity in some cases).

This medical condition is described as a specific entity in Quincke's papers from 1893 and 1897 and then by Nonne in 1904, who is the first to use the term of pseudotumor cerebri. Various hypotheses are delivered in order to explain the causes and the pathogenic mechanisms of the illness.

In 1955, Foley uses the term "benign intracranial hypertension", which is to be used for several decades, and he defines this syndrome as follows: "prolonged intracranial hypertension without ventricular modifications, without focal neurological signs, without consciousness or intellectual disorders, the most important symptoms being a moderate cephalea, blurred sight, diplopia and sometimes tinnitus. The only signs are the papillary edema and the abducens paralysis. The cerebrospinal fluid has a normal composition. The prognosis is favorable, and the symptoms progressively diminish in several weeks or months."

According to Foley's classic definition, 1955, the idiopathic intracranial hypertension occurs as a diagnosis that may be established only by exclusion; moreover, even in Dandy's diagnosis criteria, modified by Wall, one of the diagnosis elements is the non-identification of another cause for the intracranial pressure increase. The diagnosis of idiopathic intracranial hypertension can be given only after the intracranial pressure has been measured and after a complete neuro-imagistic exploration.

The diagnosis criteria of the idiopathic intracranial hypertension, which are standardized by Friedman and Jacobson, and which are currently accepted are:

- the pressure of the cerebrospinal fluid is higher than 25 cm H2O, that is higher than 18 - 20 mm Hg (by lumbar manometry performed after the CT or MRI exploration), the cerebrospinal fluid has a normal composition: normal or reduced cerebrospinal fluid proteins and normal cellularity,

- there are symptoms caused by the increased intracranial pressure: papillary edema, cephalea, with the absence of the neurological location signs (the abducens paresis is not a focal sign).

- the cerebral exploration using CT scan or MRI reveals normal cranial-cerebral aspects, a ventricular system with reduced dimensions may also be revealed (with a collapsed aspect) – without significance, or an empty sella; moreover, there is no clinical or neuro-imagistic suspicion of an intracranial venous sinus thrombosis.

These criteria limit the diagnosis of idiopathic intracranial hypertension to patients who have an increased cerebrospinal fluid pressure without a noticeable etiology.

Although it is not a diagnosis criteria, a guiding element is the predominance of the feminine sex, with a women / men proportion of approximately 2-8 / 1, with an interest in the age groups 20 - 50 years old, and with the maximum incidence in the third decade of age. [10, 11, 12, 13]

Various pathologic conditions can also be taken into account, when the idiopathic intracranial hypertension occurs more frequently: thus the global incidence of the idiopathic ICH is considered to be 1 - 2 cases / 100,000 but, in the case of young and obese women, it has been noticed that the incidence of idiopathic intracranial hypertension reaches 19-25 cases / 100,000.

The idiopathic intracranial hypertension is defined by the clinical existence of the intracranial hypertension syndrome, with very high ICP values and with a papillary edema in uncertain etiological conditions and due to pathogenic mechanisms that are difficult to establish. The term “associated factors” is used for the very diverse situations when the idiopathic intracranial hypertension occurs, without an etiopathogenic relationship.

The pathological conditions where the idiopathic intracranial hypertension occurs are numerous:

- metabolic and endocrine disorders:

- if the global incidence of intracranial hypertension is estimated at 1 - 2/100,000, in young and obese women the incidence of idiopathic intracranial hypertension reaches 19 - 25/100,000.

- hypothyroidism, parathyroid anomalies,

- hypovitaminosis A; anemia due to Fe deficiency; system illnesses – lupus eritematos;

- medicines: - nalidixic acid, tetracycline, vitamin A, lithium carbonate, etc.,

In numerous cases, a possible association can be determined with various conditions that are pathogenically suggestive, with not etiological notification; but there are plenty of cases when no etio-pathogenic relation can be revealed.

The pathogenic mechanisms are based on the dynamics of the intracranial fluids, and they correspond to the circuit of the fluids that justify the increased pressure of the cerebrospinal fluid and allow the maintenance

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of the cerebral circulatory auto-regulation with a normal nervous functioning.

The dynamics of the intracranial fluids in the case of the idiopathic intracranial hypertension can include several fluid circuits that concord to the brain edema and to the increased parenchymatous pressure, which is equal to the increased pressure of the cerebrospinal fluid, concomitantly to the maintenance of the cerebral sanguine circulation, which may explain the good clinical condition:

- hyper-production of interstitial fluid with increased resorption at the level of the brain blood barrier – which explains the increased parenchymatous pressure and the hyper-production of cerebrospinal fluid, which may explain the increased pressure of the cerebrospinal fluid, with increased resorption at the level of the venous sinuses, as well as intense and rapid exchanges from the interstitial fluid towards the cerebrospinal fluid at the level of the ventricular wall, trans-ependyma, and at the trans-cerebral pial level, which explains the pressure equilibrium between the cerebral parenchyma – ventricles, with a rapid venous drainage;

- hyper-production of cerebrospinal fluid, increased resorption at the level of venous sinuses with rapid venous drainage;

- hyper-production of interstitial fluid by modification of the brain-blood barrier, normal production of cerebrospinal fluid, but with increased exchanges from the interstitial fluid towards the trans-ependyma cerebrospinal fluid and at the trans-cerebral pial level, with a rapid venous drainage.

The increased production of cerebrospinal fluid at the level of the choroid plexuses, and then with increased resorption, cannot explain the brain edema and the pressure equilibrium at the level of the nervous parenchyma – ventricular system.

The brain edema occurs due to the impact on the brain-blood barrier, generating an increased flux of interstitial fluid, with an increase in the intra-parenchymatous pressure.

The maintenance of the cerebral sanguine circulation within normal limits imposes the decrease in the parenchymatous pressure, which can be achieved through an increased resorption of the interstitial fluid and / or by the trans-ependyma passage in ventricles, and a trans-pial passage in the sub-arachnoid space. The resulting increased quantity of cerebrospinal fluid does not cause a ventricular dilatation because an increased resorption occurs; the ventricular system is usually reduced in volume, which suggests the fact that the trans-ependyma fluid circuit is predominant for the increase in the volume of cerebrospinal fluid, compared to the production at the level of the choroid plexuses. The increased resorption of the cerebrospinal fluid corresponds quantitatively to the trans-ependyma exchange from the volume increased interstitial fluid, ensuring the intracranial circuit of fluids.

The dynamics of the relations intracranial pressure – intracranial volume is expressed by the circulation of fluids, which allows the existence of an increased fluid pressure, although this pressure does not act significantly on the endocranial structures and

on the cerebral circulation, which is maintained within normal limits.

The most probable pathogenic mechanism is the impacts on the brain blood barrier of the hyper-production of interstitial fluid and an extracellular brain edema; the cerebrospinal fluid is produced normally; there are increased exchanges from the interstitial fluid towards the cerebrospinal fluid at the trans-ependyma level and at the trans-cerebral pial level; an increased resorption of the cerebrospinal fluid is produced, and there is a rapid venous efflux.

The trans-ependyma exchange between the interstitial fluid of the edema from the cerebral parenchyma towards the cerebrospinal fluid, by means of which pressure is equalized, is followed by the increased resorption of the cerebrospinal fluid, maintaining the cerebral circulation, and representing a compensating mechanism. This mechanism is efficient and it allows the cerebral circulatory auto-regulation when there is a progressive injury of the brain-blood barrier, the interstitial fluid volume increases slowly and the brain edema is also gradually installed.

The trans-ependyma and trans-pial circuit of the interstitial fluid towards the cerebrospinal fluid is the fundamental element for this compensating mechanism of pressure increase.

The increase in the intracranial pressure is extremely slow, with a chronic aspect. This very slow increase in the intracranial pressure allows good pressure compensation and an almost normal maintenance of the cerebral sanguine flux. The pathogenic ICP values are very high, of up to 60 - 80 mm Hg, values that may be maintained on a plateau for long periods.

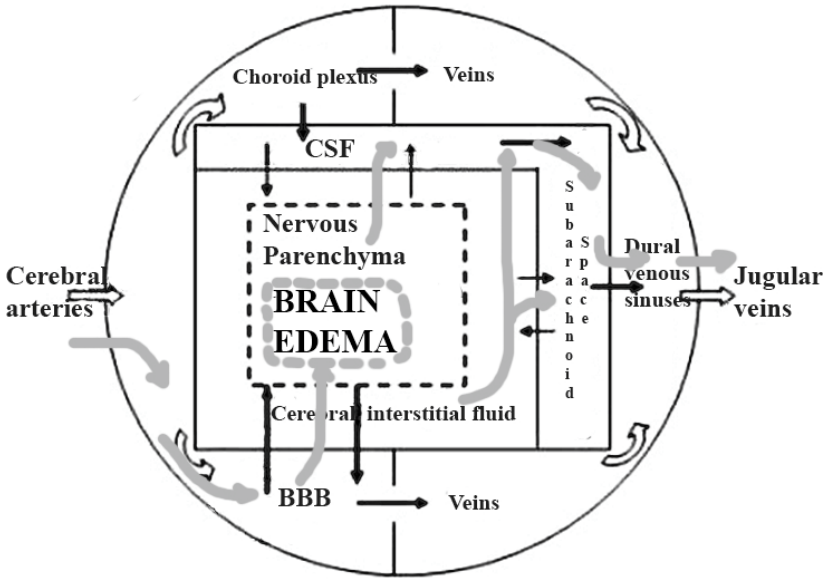

Figure 1 - Hydrodynamic model of pathogeny in idiopathic intracranial hypertension

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This pathogenic mechanism, which is based on the compensation of the fluid pressure increase by means of a gradual accentuation of the trans-ependyma circuit functioning and the trans-pial transfer of the interstitial fluid towards the cerebrospinal fluid, explains:

- the absence of hydrocephalus despite the increased pressure of the cerebrospinal fluid, through increased fluid resorption,
- the small dimensions of the ventricular system due to the initially increased pressure at the level of the nervous parenchyma, and subsequently balanced by the transfer of the interstitial fluid towards the cerebrospinal fluid,
- the good clinical condition due to the auto-regulation of the cerebral circulation, because the increased intracranial pressure has a reduced action on the endocranial structures in the dynamic conditions of the rapid fluid circuit;
- the brain edema frequently exists due to the dysfunction of the brain blood barrier and to the existence of an increased quantity of interstitial fluid; at the same time, in some cases, the brain edema may be reduced or even absent because the fluid transfer towards the cerebrospinal fluid is sufficiently rapid and intense for the brain edema not to be significant.

Treatment: the therapy is applied depending on the possible associated factors – metabolic and hormonal corrections, exclusion of certain medication, etc. A symptomatic treatment is applied for cephalea, and cerebral anti-edematous substances are administered, such as dexamethasone, or diuretics (acetazolamide, furosemide) in order to decrease the intracranial pressure. In some cases of obese patients, the weight loss is followed by an obvious clinical improvement, and by the progressive reduction of the papillary edema. Moreover, repeated lumbar punctures and lumbar-peritoneal shunting have been used with good clinical results. An approximate quantity of 30 ml CSF is evacuated by lumbar puncture or the cerebrospinal fluid is drained until the intracranial pressure, which is measured by the lumbar method, is seen to have decreased to approximately half of the initial value. The lumbar peritoneal shunting drains the excessive CSF and the symptomatology disappears within less than a month; the shunting revising rate may reach to 50 %. In some cases of significant visual acuity decrease, there has been an attempt to decompress the optic nerve by performing fenestrations at the level of the optic nerve sheath in order to produce a decrease of the papillary edema, but the results are not always favorable.

Comparison between idiopathic ICH and the vascular type of ICH

The recent literature data and the the above presentations allow the differentiation between the vascular type of ICH and idiopathic ICH. The name hyperemic ICH has been suggested but the designation of vascular ICH covers the etiology and pathogenesis better and more completely.

Although the symptoms may be similar in these two diseases, the imaging findings, therapy and evolution are different; therefore,

the etiology and pathogenesis are different. Vascular ICH has a known etiology, such as cerebral vascular illness whereas idiopathic ICH has no known etiology or has various nonspecific factors associated factors (e.g., metabolic and endocrine disorders, hypovitaminosis A, and medications). The imaging diagnosis for vascular ICH showed a cerebral venous sinus thrombosis or stenosis or a cerebral venous thrombosis and either normal images or small ventricles in idiopathic ICH. The imaging diagnosis of idiopathic ICH excludes other diseases with similar symptoms.

Vascular ICH involves vasogenic brain edema with papillary edema, whereas idiopathic ICH commonly involves papillary edema and diminished visual acuity in some cases. There is also brain edema in idiopathic ICH, but this appears to be balanced by the intraventricular pressure. The increase in ICP is faster in vascular ICH compared with the very slow ICP increase in idiopathic ICH, as demonstrated by the longer period until the complete clinical syndrome has developed. In addition, the ICP values are higher in idiopathic ICH. Therefore, the critical ICP values are lower in vascular ICH until the decompensation of ICH.

The treatment is symptomatic, as well as etiologic and pathogenic in vascular ICH but only symptomatic in idiopathic ICH (including a lumboperitoneal shunt or decompression of the optic nerve).

Vascular ICH may potentially include other syndromes, such as hypertensive encephalopathy, but this classification requires further study. In hypertensive encephalopathy, there is a dilatation of brain arteries with increased arterial inflow, increased vascular permeability, and disruption of the braineblood barrier; brain edema occurs as a result.

In conclusion, ICH caused by intracranial vascular damage is vascular ICH and although it has clinical similarities to idiopathic ICH, there are important differences: vascular ICH has a known etiology, such as cerebral vascular illness, and the increase in ICP is faster in vascular ICH, but the critical ICP values are lower compared with idiopathic ICH.

The therapy is etiologic, pathogenic and symptomatic in vascular ICH, but is only symptomatic in idiopathic ICH.

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